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Apolipoprotein A-IV levels and phenotype distribution in NIDDM.

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OBJECTIVE--To determine plasma apolipoprotein A-IV (apoA-IV) levels and phenotype distribution in non-insulin-dependent diabetes mellitus (NIDDM) patients and to analyze the influence of apoA-IV phenotype on lipid profiles in NIDDM. RESEARCH DESIGN AND METHODS--Total cholesterol, triglyceride, high-density lipoprotein (HDL) cholesterol, HDL2 cholesterol, HDL3 cholesterol, free fatty acid, and apoA-IV levels were measured in 83 NIDDM patients and 100 normal control subjects. The apoA-IV phenotype was determined in each individual. RESULTS--In both sexes, NIDDM patients had significantly higher levels of triglyceride and free fatty acid and significantly lower levels of HDL cholesterol and HDL2 cholesterol than control subjects. In men and women, apoA-IV levels were significantly higher in diabetic patients than in control subjects (men: 17.1 +/- 7.9 vs. 12.3 +/- 3.6 mg/dl, $P < 0.001$; women: 18.9 +/- 9.9 vs. 11.9 +/- 3.5 mg/dl, $P < 0.001$). The multiple regression analysis showed that the apoA-IV level in NIDDM patients was significantly and independently related to log triglyceride ($P = 0.0001$) and HDL cholesterol ($P = 0.01$) levels. The apoA-IV phenotype distribution was not significantly different between NIDDM patients and control subjects. In the control subjects, the apoA-IV-1-2 phenotype was associated with significantly higher levels of HDL cholesterol (69 +/- 12 vs. 56 +/- 11 mg/dl, $P < 0.01$) and of HDL2 cholesterol (36 +/- 15 vs. 25 +/- 12 mg/dl, $P < 0.05$) compared with the apoA-IV-1-1 phenotype; on the other hand, HDL cholesterol and HDL2 cholesterol levels were not different between the two apoA-IV phenotypes in NIDDM patients. CONCLUSIONS--Plasma apoA-IV levels are increased in NIDDM patients. This increase in apoA-IV is related mainly to hypertriglyceridemia and, to a lesser extent, to HDL cholesterol level. The apoA-IV phenotype distribution is not different between NIDDM patients and nondiabetic control subjects. The potential protective lipid profile (characterized by increased HDL and HDL2 cholesterol levels) linked with

apoA-IV-1-2 phenotype in control subjects is no longer found in NIDDM patients. We suggest that the metabolic state of NIDDM has erased the potential protective lipid profile associated with the apoA-IV-1-2 phenotype.

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